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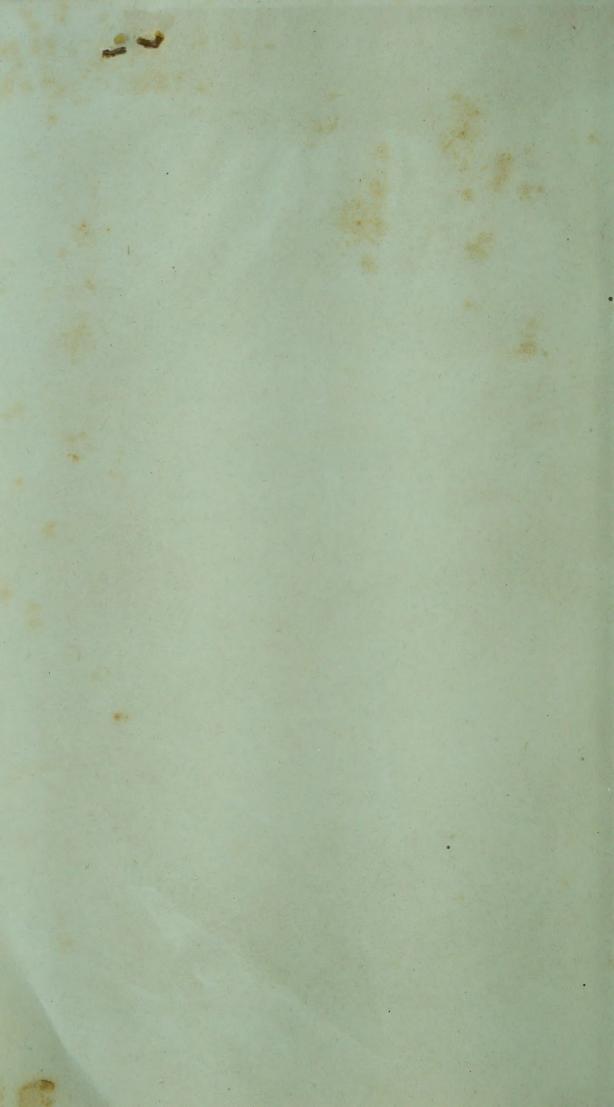
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Notes on Communicable Diseases of Laboratory Animals

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NOTES ON COMMUNICABLE DISEASES OF LABORATORY ANIMALS

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PREFACE

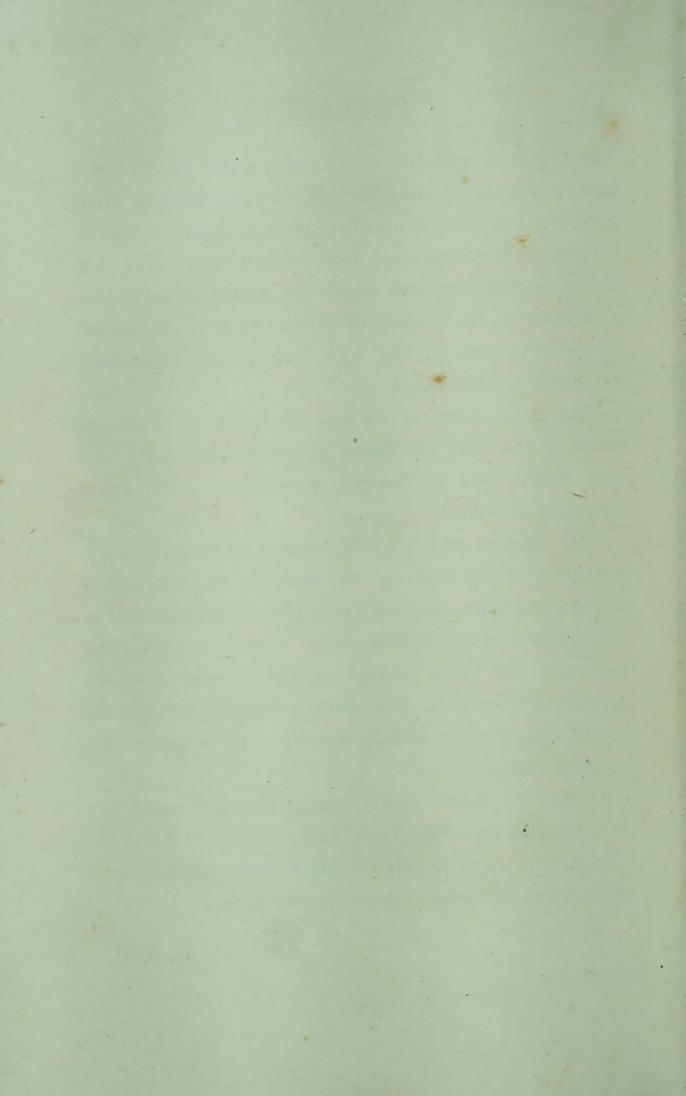
THESE general and not too detailed notes on the contagious diseases of laboratory animals may help to make good the lack of a convenient short monograph on the subject. They originated from several suggestions that the cyclostyled synopsis which the author circulated at a meeting of the Society of Public Analysts (Biological Methods Group) on 21st April 1947 should be revised, extended, and published.

The more common diseases receive most attention. Some of the rarer conditions are mentioned in the text and index only to suggest possibilities when exact diagnosis is difficult.

The general principles of control of infections are described at the ends of the sections on guinea-pigs, rabbits, and mice, and will be helpful to all interested in the maintenance of healthy stock. Certain modifications necessary for other animal species are indicated in appropriate sections, but more complete information would sometimes be largely repetitive and is outside the scope of this booklet.

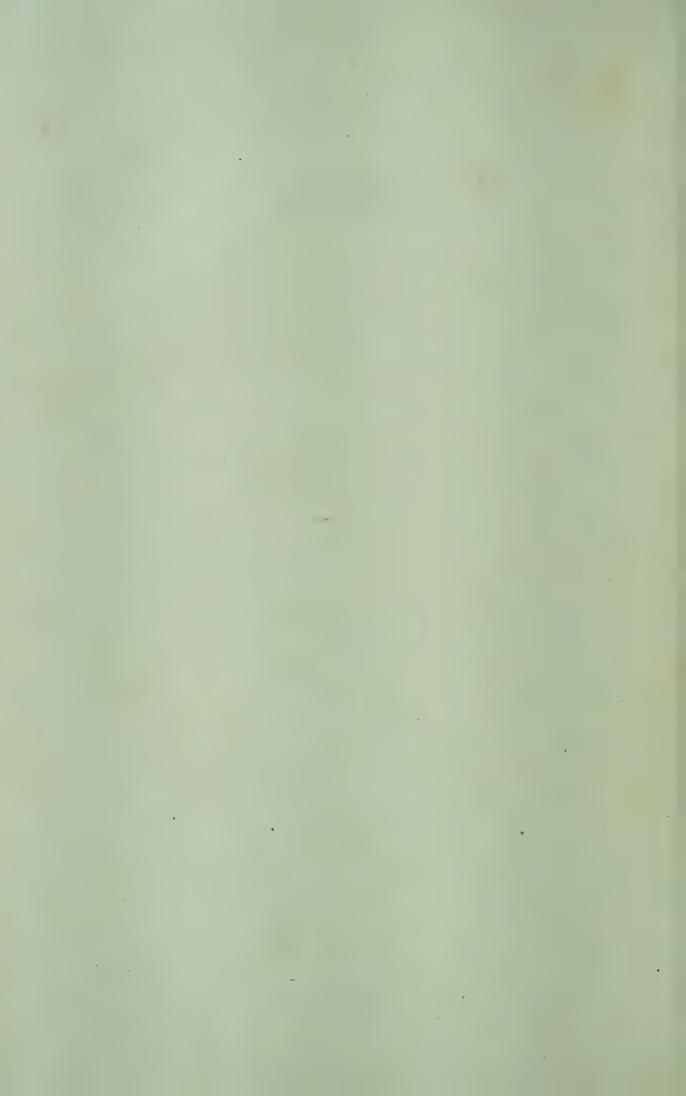
I wish to thank many friends and colleagues for valuable data, especially Mr A. B. MacIntyre, M.R.C.V.S., who gave me considerable assistance in the preparation of the sections on ferrets, cats, dogs, and pigeons.

H. J. P.



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GUINEA-PIGS

RESPIRATORY INFECTIONS. Common. Sporadic cases and severe epizootics.

Symptoms.—Rather indefinite.

Nasal discharge (watery)

Coughing

Sneezing

Cervical glands:

Frequent.

Sometimes enlarged.

Post-mortem.—Changes variable.

Pleurisy:

Serofibrinous exudate, with ad-

hesions.

Pneumonia:

Diffuse or patchy.

Pericarditis 7

Less common.

Peritonitis

Hæmorrhages and

septicæmia:

Frequent.

Causal Organisms.-

Pneumococcus.*

Pneumobacillus.

Streptococcus.

Pasteurella group.

Hæmophilus bronchisepticus.

* The pneumococcus may also cause uterine infection in pregnancy. Either living fœtuses and early death of mother, or dead fœtuses.

Post-mortem.—

Uterus and appendages: Inflamed.

Peritonitis: Liver:

Frequent. Pale from fatty degeneration.

(In uterine type of pneumococcal infection, thoracic organs relatively normal.)

HÆMOLYTIC STREPTOCOCCAL INFECTIONS.

Common.

Bubonic Type.—Chronic abscesses in lymph glands. A special type of *cervical adenitis* due to Group C streptococcus is described. Entry of infection probably by damaged buccal mucosa.

Septicæmic Type.—Rapidly fatal.

Intermediate Types.—Numerous. Inflammatory changes, with or without necrotic patches or abscesses in one or more of the following organs or tissues:—

Lymph glands.

Lungs: Pneumonia (often hepatisation), with

copious hæmorrhagic pleural effusion.

Heart: Epizootic type also described with non-

purulent carditis and valvulitis. Some-

times pericarditis with hæmorrhages.

Liver.

Spleen.

Intestines: Almost always bright yellow in colour.

Mammary glands.

Muscles.

Subcutaneous

tissues: In extreme case, extensive inflammation

over abdominal and thoracic regions

(? related to injections).

Joints: Arthritis has been described.

CAUSAL ORGANISMS.—Isolated from blood or pleural effusion, etc. Almost always Lancefield Group C.

SALMONELLA INFECTIONS. Common. Sometimes called guinea-pig plague or "paratyphoid."

SYMPTOMS.—

Staring coat.

" Arched" back.

Weakness.

Wasting:

Rapid or gradual.

Diarrhœa:

Not marked in natural disease.

Mortality.—Four to 70 per cent in different epizootics. Death after two to three days (acute cases), or many weeks (chronic variety).

Post-mortem.—In acute cases, macroscopic lesions usually slight; possibly only slight enlargement of spleen. In chronic cases, more marked, as follows:—

Gall-bladder:

Very often contains pus.

Intestines:

Congested and possibly ulcerated.

Mesenteric lymph

glands:

Always enlarged and œdematous.

Spleen \

Enlarged and often studded with greyish

Liver 5

areas or abscesses.

Peritoneum:

Sometimes peritonitis, with thick exudate over liver, spleen, etc. Peritonitis may

be general or local

be general or local.

Uterus:

Occasionally abscesses.

CAUSAL ORGANISMS.—Salmonella group, e.g., S. typhimurium (B. ærtrycke). Less commonly S. enteritidis (B. gaertner) and the Reading type.

Isolation from :—

Spleen

Liver and gall-bladder \Usually easy.

Heart blood

Stomach and intestines (aid of brilliant green).

Mesenteric glands, etc.

Sometimes infection precipitated in apparently healthy stocks by injudicious feeding (frosted greens, mouldy oats, too many mangolds, etc.).

Guinea-pigs which recover may become carriers, and initiate

a fresh outbreak in another colony.

PSEUDO-TUBERCULOSIS. Often confused with tuberculosis.

Acute or Septicæmic.—

Staring coat.

Respiratory symptoms, e.g., coughing and rapid breathing. Death in a few days.

Chronic or Subacute: Glandular type.—

Diarrhœa.

Wasting.

Palpable nodules: Sometimes.

Duration possibly several weeks.

POST-MORTEM.—

Acute Type.—Changes usually indefinite as disease rapidly fatal.

Lungs: Congested.

Chronic Type.—

Mesenteric glands

Spleen

Liver

Numerous creamy or caseous yellowish - white nodules (abscesses). Well defined, and often protrude from surface.

Small intestine: Sometimes abscesses of Peyer's

patches.

Peritoneum: Sometimes seropurulent peritonitis, with adhesions to

liver, etc.

Lungs

Pleuræ

Pericardium

Subcutaneous tissues

Sometimes inflamed and may contain nodules.

CAUSAL ORGANISMS.—

Pasteurella pseudo-

tuberculosis: Readily isolated from lesions.

P. septica: Much less common. Causes sero-

purulent inflammation of peritoneum, pleuræ, and pericardium.

Certain other bacteria may cause pseudo-tuberculous lesions from time to time.

Transmission is probably by ingestion.

Mothers may infect their offspring shortly after parturition.

MISCELLANEOUS INFECTIONS, etc.

Infection with *Pseudomonas caviæ* (capsulated Gramnegative bacillus isolated from blood and viscera).

Epizootic septicæmia of young guinea-pigs.

Eyelids stuck together.

Lymph glands: Enlarged.

Listlessness.

Prostration.

Dyspnœa.

Death in five to six hours.

Cervical adenitis due to Streptobacillus moniliformis or Actinomyces muris.

Cervical abscesses, causing large swellings.

Thick creamy pus: Dense fibrous capsule.

Little or no effect on health.

Lesions often regress or, alternatively, rupture externally.

DIFFERENTIAL DIAGNOSIS.—Mainly from pseudo-tuberculosis and a cervical adenitis due to Group C streptococci.

Coccidiosis.—Uncommon in the guinea-pig although common in the rabbit.

Diarrhœa with passage of mucus may occur, although symptoms usually absent.

If death occurs, usually associated infection.

Post-mortem.—Yellow nodules in liver and intestines. However, tissue damage may be slight, even when gut heavily infected.

CAUSAL ORGANISM.—Only one species of coccidium, viz., Eimeria caviæ.

Virus Diseases.—Several described, including:—

1. Guinea-pig paralysis.

2. "Salivary virus" disease of Cole and Kuttner: Unimportant.

No symptoms in naturally infected stock.

Latent virus infections may cause confusion and error when guinea-pigs are inoculated experimentally with other viruses.

LICE.—Treat with D.D.T. or Gammexane

Tumours.—Rare.

Sarcoma: Slow-growing

Adenoma

Lipoma

Outside the scope of these notes, but probably worthy of mention.

CONTROL OF INFECTIONS IN GUINEA-PIGS

Reliable, conscientious assistants, and suitable rooms and sterilising equipment are indispensable in all animal work.

Natural infection is mostly by digestive tract, but also respiratory spread in certain diseases.

Source.—Cases and carriers (e.g., guinea-pigs with pathogenic organisms in fæces, but no symptoms).

Injudicious feeding, bad housing, etc., may light up infection.

Only authorised persons should be permitted to enter animal rooms. Stray dogs and cats, and also wild rats and wild mice excluded, as they may introduce dangerous bacteria and parasites.

Once infection introduced, epizootic often kept going by overcrowded conditions and by mixing stocks.

Sick animals are best destroyed (few exceptions).

Emphasis on prevention--not on treatment.

Post-mortem examinations, with cultures, of all carcases: the ideal procedure. Seldom followed in practice, although may help in forestalling a dangerous epizootic.

A death-rate above 2 per cent per week should be regarded

as unsatisfactory.

Cages.—Dry, light, well-ventilated, and scrupulously clean. Wire-mesh floor, with shelf underneath for tray (emptied daily and sterilised).

Wooden cages and racks are unhygienic and should be

scrapped.

Modern type of *cage trolley* facilitates movement with little disturbance.

Runs.—Paper, with bedding on top, has been used with excellent results. Cleaning facilitated. A satisfactory alternative to cages.

Adequate hot water, soap, and stiff-bristled brushes of suitable shapes are necessary for thorough cleansing of cages, etc.

Frequent disinfection of cages in cage-autoclaves or possibly in tanks.

Hosing down of runs and floors.

Lysol or cresols should be used with care as they may cause irritation of the mucous membranes. Preparations of the dettol type might be preferable.

Carrying boxes and crates should also be scrubbed and disinfected.

Bedding (from runs) removed twice weekly and burned.

Fresh, dry, and clean (uncontaminated) bedding: preferably stored in bins or metal containers in special rooms protected from vermin and stray rodents. At many laboratories all incoming bedding sterilised as routine—much to commend this practice. Temperature and humidity reasonably uniform. Sudden variations harmful.

Cold spell, with inadequate artificial heating, may cause increase in death-rate.

Thermostatically controlled heating and air-conditioned ventilation considered when new quarters erected.

Draughts should be avoided.

Food and Water.—Metal or earthenware pots or bowls scalded daily. Placed above floor level. Of type not readily contaminated by animals' feet or fæces.

Food: Stored in metal contained in rooms set aside for purpose. Not spilt on floors.

Vermin and stray rodents excluded from building.

Regular feeding. Proper food. Fresh *Brassica* (preferably cabbage) important.

Clean (and fresh) water. Water bulbs less easily contaminated than open vessels.

Isolation of New Stock.—Ten to fourteen days. Longer (up to two months) if disease occurs.

Isolation of animals on basis of agglutination or other blood test has failed to control spread of infection.

Immediate Destruction of Ailing Animals.—Burn carcases.

Tray with Mat and Disinfectant.—Outside door of animal room. Reduces risk of transfer of infection.

Specific Prophylaxis and Treatment.—Sera and vaccines useless. Sometimes apparent success for limited period, but failure to repeat success. (Other factors involved in first instance.) Considerable trouble and expense.

Chemotherapy.—Addition of sulphonamide to diet for short period has been suggested and may be useful for some infections. Results difficult to interpret. Other drugs may be tried.

RABBITS

COCCIDIOSIS. Probably commonest and most serious disease of rabbits. Hepatic and intestinal forms described.

Acute.—

No previous ill-health.

Death sudden (70 to 100 per cent of litter may die).

Subacute.—

Diarrhœa and ravenous appetite: Common symptoms. Coma and convulsions, soon followed by death.

Chronic (in adults).—

Diarrhœa.

Progressive emaciation.

Pot-belly.

Weakness of hind legs.

Carriers.—Frequent in adult stock. Play important part in spreading infection.

POST-MORTEM.—

Acute Type.—

Indefinite.

Oöcysts of parasite demonstrated in liver preparations.

Other Types.—Principal lesions in intestines and liver :—

Intestine: Typically, multiple white nodules.

Liver: Often multiple white or yellowish nodules.

Bile ducts sometimes seen as irregular white streaks on surface of liver. Liver may be much enlarged from pressure on bile ducts. In young rabbits possibly a fatal hepatitis, due to multiplication of the

parasite (Coccidium) in the bile duct

epithelium.

Causal Organisms.—Two forms of Coccidium, viz.:—

- 1. Eimeria stiedæ.—Causes the liver or hepatic form.
- 2. E. perforans.—Causes the intestinal form.

Oöcysts are passed with the fæces in both forms, and can be demonstrated microscopically.

Are not infective for about two days after being passed. (The actual period varies somewhat with humidity and temperature.)

An oöcyst count is an unreliable index of the severity of infestation.

CONTROL.—

Remember that many adult rabbits are carriers.

As treatment is difficult, rely on prophylaxis.

Weed out infected breeding does.

Timely weaning and segregation of litters from parent does will help.

All sick animals immediately destroyed and carcases burned. Advisable, in fact, to kill whole litter, as survivors are potential carriers. Seldom carried out.

Rabbits kept in cages, on wire-mesh floors, with tray underneath for droppings.

If on grass out of doors, move on to clean grass every two days.

Attention to hygiene and housing, including disinfection of cages and crates (blowlamp applied): warm, weak, commercial or household ammonia has been recommended. Unspillable feeding-troughs and drinking vessels desirable.

Hay and all green food infected by wild rabbits avoided as prophylactic measure.

Chemotherapy, e.g., mash containing 1 per cent sulphamezathine, or drug in saturated solution in drinking water.

SALMONELLA INFECTIONS. An intestinal infection less common than in guinea-pigs. Few references in the literature.

SYMPTOMS.—

Diarrhœa the main symptom.

Accelerated respiration, rise of temperature, and loss of appetite also noted.

CAUSAL ORGANISMS.—Salmonella group, e.g.:

S. typhimurium (B. ærtrycke).

S. enteritidis (B. gaertner): Recovered rarely.

May be isolated from blood-stream and various internal organs.

"SNUFFLES." Most common of respiratory infections.
Usually chronic. A frequent source of trouble, as it is highly contagious.

SYMPTOMS.—

Nasal discharge: Serous or seropurulent (persistent, white, tenacious, copious). Dried crust of exudate around nostrils.

Sneezing: Often the first symptom.

Tendency to rub nose on paws.

Head shaking: May be due to ear infection.

Anorexia.

Coughing

Weakness \rangle In chronic types.

Wasting Coat staring

Coat staring: Fur may become soiled and matted

with discharge.

Death:

(Often from pneumonia and pleurisy.

May be septicæmia.) Occurs after
a few days in acute forms, with
complications. In the chronic disease
ultimate mortality rate also high.

Carriers.—Infected animals which recover. If resistance lowered, a further attack.

Post-mortem.—

Rhinitis: Purulent. Sinusitis: Frequent.

Otitis
Meningitis
Occasional.

Lungs: Bilateral confluent broncho-pneumonia.

Pleuræ: Sometimes exudate.

Pericardium: May be involved in general inflammatory

process.

Peritoneum: Sometimes peritonitis, without an associ-

ated pleuro-pneumonia.

CAUSAL ORGANISMS.—

Pasteurella lepiseptica (P. cuniculicida) and Hæmophilus bronchisepticus occur singly or together in discharge and lesions.

(P. lepiseptica belongs to "hæmorrhagic septicæmia" group of bacteria in different animals, and may cause a terminal septicæmia in "snuffles." H. bronchisepticus may be only a secondary invader.)

Also found in air-passages of "normal" rabbits (carriers).

CONTROL.—

Infected animals destroyed at once before infection spreads. More profitable than isolation. (Experts differ; some advise isolation.)

Cages, crates, etc., disinfected.

Improve housing and feeding.

Chemotherapy: Local or general. Infection appears to be prolonged, but seldom cured.

PSEUDO-TUBERCULOSIS. Often confused with tuberculosis. Is frequent in wild rodents. Acquired in animal houses by ingestion of contaminated food and other material.

SYMPTOMS.—

Usually chronic.

Often unnoticed in early stages.

Wasting.

Laboured respiration.

Weakness.

Death usually in three to four weeks.

POST-MORTEM.—

Lungs

Liver

Spleen

Kidneys

Intestines

Mesenteric lymphatic

glands.

Joints:

Numerous small cheesy nodules in any or all of these organs.

Subcutaneous tissues: Sometimes a large abscess.

May be lesions in very advanced

cases.

CAUSAL ORGANISM.—

P. pseudo-tuberculosis: Easily isolated in more acute cases. More difficulty in chronic cases.

TUBERCULOSIS. Infrequent as a rule.

Usually bovine type. Acquired from milk from tuberculous cows, or possibly from attendant with tuberculosis.

Avian type infrequent. Due mostly to food contaminated by droppings of birds.

SYMPTOMS.

Acute Miliary Type.—No symptoms till a few days before death.

Chronic Type.—

Wasting for two to three weeks.

Loss of appetite.

Weakness.

Diarrhœa.

Laboured respiration develops before death.

POST-MORTEM.—

Numerous irregular tubercles in most internal organs. Caseous lesions in associated lymphatic glands. Acid-fast bacilli in films.

ULCERATIVE LESIONS OF GENITALS, VENT DISEASE or RABBIT SYPHILIS. Common. A true venereal disease not transmissible to other animal species or man.

Symptoms.—

Genitals: Small scaly sores or papules, often with slight erosions and brown crusts. Size and severity variable. Sometimes swelling, especially in the doe, which may cause difficulty in passing urine and fæces.

Secondary lesions frequent on mucous membranes of lips and nose, and on conjunctiva and cornea. No generalisation to internal organs as a rule.

Unless generalisation, health reasonably good and death unlikely.

CAUSAL ORGANISM.—

Treponema cuniculi: Somewhat larger than T. pallidum. Present in sores. Transmitted during mating.

CONTROL.—

No breeding using infected animals.

Animals required for breeding carefully inspected before transfer to sterilised cages or hutches.

Treatment with mercurial ointments locally or arsenicals intravenously very successful. One injection usually cures.

Penicillin and other antibiotics.

MANGE. Commonest parasitic infection in the rabbit. Contagious directly or by handling. Two types:—

Ear Mange or Ear Canker.—

External auditory meatus affected.

Ear swollen at base.

Inner aspect of ear covered with scabs.

Middle and inner ear may be affected secondarily.

Infected rabbit scratches ears with hind claws and shakes head.

Body or Skin Mange.—

Loss of areas of fur.

Scab formation on roots of ears, face, and head.

Considerable irritation, leading to emaciation and death.

CAUSAL PARASITES.—

Both types caused by two kinds of mite.

Ear mange is due to *Psoroptes communis cuniculi* or *Chorioptes cuniculi*. (Symptoms similar, but chorioptic mange is usually less severe.)

Body mange is caused by Sarcoptes cuniculi and Notöedres cuniculi.

These mites readily demonstrated in wet smears of scab treated with 10 per cent caustic potash solution.

CONTROL.

Easily treated.

Liquid paraffin containing 1 per cent phenol.

Sulphur
Balsam of tolu
Carbolic oil
Probably less effective. (Sulphur may cause dermatitis on hand of attendants.)

Benzyl benzoate preparations, Tetmosol, and also Gammexane: Recently suggested.

Hydrogen peroxide: Sometimes used to remove scabs.
All cages of infected rabbits must be thoroughly disinfected.

MISCELLANEOUS INFECTIONS, ETC.

schmorl's disease or Necrobacillosis. Ulcerative condition. Infectious. Uncommon in domesticated rabbit. Tends to be chronic, and subcutaneous swellings may give clue to diagnosis.

SYMPTOMS.—

Skin: Ulceration and necrosis, especially of head and neck. "Labial necrosis."

Abscesses: Thickly walled, in different parts of body (subcutaneous tissues, lungs, liver, and kidneys).

Jaws: Moved with difficulty if neighbouring lesion.

Pneumonia May develop.
Pleurisy

Progressive emaciation and death.

CAUSAL ORGANISM.—

Fusiformis necrophorus.—Rod-shaped organisms of varying length, usually Gram-negative. Belongs to normal skin flora of rabbit, and invades blood-stream via skin wounds or alimentary tract.

A ruptured subcutaneous abscess may spread infection to other rabbits via wounds caused during fighting or otherwise.

CONTROL.—

Cleansing and antiseptic treatment of skin wounds suggested.

Penicillin may be tried.

Once abscesses have developed, kill the animal.

STRANGLES.*

SYMPTOMS.—

Region of lower jaw.

Variable size (sometimes as large as tennis ball).

Firm to touch.

Contents are thick greyish-yellow pus.

Abscess \{\) No tendency to burst spontaneously.

Often extension to bones of jaw and also

surrounding tissues.

If neglected, vital structures involved, resulting in death.

CAUSAL ORGANISM.—

Streptococcus.

Similar condition may be caused by other pyogenic bacteria, but this is not called *strangles*.

Fusiformis necrophorus may invade abscess secondarily.

CONTROL.—

Open abscesses surgically.

Remove contents.

Usual hygienic measures.

MASTITIS and PYOMETRA. Sporadic inflammatory conditions of udder and teats, causing interference with suckling, and of the uterus, leading to sterility.

SYMPTOMS.—Chronic purulent discharges, from which a variety of pyogenic bacteria and sometimes *F. necrophorus* may be isolated.

CONTROL.

Mild antiseptic ointments and uterine douches may be tried.

Stilbæstrol.

^{*} Misuse of the specific name for a disease of horses.

VIRUS DISEASES. The following are included:—

I. Virus III of Miller, Andrewes, and Swift is the commonest.

Acute orchitis.

Readily transmitted.

2. Myxomatosis.—

Found in America.

Multiple subcutaneous tumours, which are gelatinous and vascular.

Conjunctivitis an early symptom.

Orchitis has occurred.

Disease usually fatal in two to five days.

3. Rabbit-pox.—

Skin eruption.

High temperature and prostration.

Mortality high in young rabbits (about 70 per cent).

Highly contagious.

Virus related to vaccinia virus.

TRYPANOSOME INFECTIONS. No apparent symptoms.

Causal Organism.—Trypanosoma lewisii. Infection is a hutch one transmitted by the rabbit flea.

SPONTANEOUS ENCEPHALITIS. Occurs in Europe and America.

Post-mortem.—Lesion of granulomatous type.

Causal Parasites.—Two described, viz., Encephalitozoon cuniculi and Toxoplasma cuniculi.

FAVUS and RINGWORM. Uncommon. Appearances and treatment as in man.

WORMS and other INTERNAL PARASITES. Unimportant.

Cysts { Usually one only, causing subcutaneous swelling. May be multiple—omentum and liver. Clear contents.

CAUSAL PARASITES.—

The commonest is *Cysticercus pisiformis*, the cysticercus or larval stage of *Tænia pisiformis* (*T. serrata*) of dogs. Liver flukes: Uncommon.

CONTROL.

Subcutaneous cyst can be removed surgically and destroyed.

Dogs should not have access to food-stuffs or drinkingwater of rabbits.

TUMOURS. Rare.

Fibroma Lipoma Sarcoma

Slow-growing connective tissue tumours.

Carcinoma: Even more rare. A rapid-growing carcinoma has been described.

CONTROL OF INFECTIONS IN RABBITS

Except where specified in these notes, *treatment* is difficult and usually unprofitable.

Reliance is placed on prophylaxis.

As in the case of guinea-pigs, infections may be kept going by unhealthy conditions. Overcrowding should be avoided. Single cages best. Injudicious feeding, etc., may light up infection.

Post-mortem examinations, with cultures, of all carcases the ideal procedure.

Cages.—Dry, light, well-ventilated but not draughty, clean. Wire type good. Wire-mesh floor, with shallow shelf containing tray underneath (emptied daily and sterilised).

Cage replaced weekly. Cage autoclaves or tanks essential

for disinfection of cages after thorough scrubbing.

Lysol or cresols should be used with care as they may cause irritation of mucous membranes. Preparations of the dettol type might be preferable.

Carrying boxes and crates should also be scrubbed and

disinfected.

Bedding.—From cages with trays: Trays emptied daily and contents burned. From cages without trays: Removed twice weekly and burned.

Can use paper underneath shavings for convenience in cleaning.

Hard wood (teak, mahogany, etc.) shavings or sawdust may be harmful, due to phenolic substances.

Fresh bedding best stored in bins or metal containers in special rooms protected from vermin and stray rodents.

Food and Water.—Containers of metal or earthenware, scalded daily. Placed above floor level to exclude unnecessary contamination.

Feed regularly with proper food. (Overfeeding after missed meals: Indigestion caused, and latent infections lit up.)

Food, like bedding, stored in bins or metal receptacles in store-rooms inaccessible to vermin and stray rodents. Should be good, clean, and not frozen—a point sometimes overlooked.

Clean (and fresh) water supplied in water-tins hung on

inside of cage doors.

Water bulbs preferable, as more difficult to foul.

Note that animals which have been bled require fluid for replacement, and should have access to plenty of drinking water.

Isolation of New Stock.—Period of two weeks suggested—longer if disease occurs.

Hutches and hutch utensils should be thoroughly disinfected before fresh animals transferred to new quarters.

Immediate Destruction of Ailing Animals.—Carcases burned after autopsy.

Consider destruction of litter mates, e.g., in coccidiosis.

Destruction of all Animals in Batch.—May be necessary if epizootic extensive.

Chemotherapy.—A limited field of usefulness in special circumstances.

MICE

INTESTINAL INFECTIONS or "MOUSE TYPHOID."

Common.

Very variable course : [Influenced by many variables, e.g. :

Acute Subacute Infecting dose or doses. Virulence of organisms.

Chronic

Resistance of strain of mice.

Resistance of individual mouse.

Incubation period:

Variable

Age of animal. Season of year.

Three days and Temperature.

upwards

Diet.

Infection usually by oral route. Introduced by carriers or contaminated food.

Symptoms.—

Mouse looks ill.

Hair roughened.

Usually present.

Loss of appetite.

Loss of weight:

In chronic cases, pronounced.

Conjunctivitis:

May develop. Eyelids glued together.

Respiration:

Diarrhœa *:

Accelerated.

In acute cases death in about seven days.

Post-mortem.-

General congestion of blood-vessels and viscera.

Intestines:

Redness, injection, and swelling of mucous membrane, possibly with ulceration.

Liver Spleen Enlarged and usually dark red. contain grey necrotic lesions. Changes variable and splenic enlargement may be only obvious sign of infection.

^{*} It should be noted that diarrhoea is not necessarily a symptom of "mouse typhoid." It may be due to some non-infective cause, viz., diet deficient or otherwise faulty.

MICE 23

Peritoneum:

Sometimes serosanguineous exudate, and there may be adhesions between coils of intestines.

Lymph nodes <

Slightly or moderately enlarged. This enlargement may be general, and chronic Salmonella infections have been mistaken for leukæmia on basis of enlarged glands.

Lungs:

May be normal or show pin-point hæmorrhages or congestion.

CAUSAL ORGANISMS.—

Salmonella group: S. typhimurium (B. ærtrycke), and, less commonly, S. enteritidis (B. gaertner).

Readily isolated from blood-stream and various organs.

Isolated also from fæces of carriers.

Bacteriological findings establish the diagnosis.

CONTROL.—

Same as for various infections in guinea-pigs and rabbits.

If highly fatal epizootic, advisable to destroy all infected stock, and sometimes entire herd.

Carcases burned.

Boxes disinfected.

Fresh stock obtained.

Killed vaccines not effective in prevention.

Repeated cultures to detect and exclude carriers very expensive and not very helpful.

Survivors of an epizootic often carry the organisms.

In paying attention to hygiene and housing, inquire into possible contamination of food by wild mice and other wild rodents, and by flies.

Hands of attendants may transfer infection during cleaning, etc. Frequent washing would eliminate risk, but cannot be enforced in practice.

"SEPTICÆMIC DISEASES" OF MICE. Apart from pasteurellosis, unimportant.

Much confusion in terminology. The following three main types are described (note the overlap):—

1. Pasteurellosis.—

So-called "hæmorrhagic septicæmia" group.

Mainly due to Pasteurella muriseptica or P. muricida (B. murisepticus).

Pseudo-tuberculosis due to *P. pseudo-tuberculosis* is also included in this group; it is rare in mice.

2. Pseudo-tuberculosis.—

The term is used loosely to include many diseases of varied ætiology. Thus one type is included in the previous paragraph.

Salmonella organisms cause a second type (see section on Intestinal Infections or "Mouse Typhoid").

The third and main variety is caused by Coryne-bacterium pseudo-tuberculosis (C. kutscheri, Bergey), a diphtheroid of specific pathogenicity for mice. May get mild epizootic spread, acute or chronic, with signs of enteritis.

Relatively frequent.

3. "Mouse Septicæmia."—

Due to *Erysipelothrix muriseptica* (short Gram-positive rods and, in culture, longer and filamentous forms), allied to *E. rhusiopathiæ*, the causal organism of swine erysipelas.

All these diseases generally acute; insufficient time for characteristic morbid changes.

In less acute cases the signs are somewhat similar to those described under pseudo-tuberculosis (pasteurellosis) of guineapigs and rabbits. The gross lesions suggest tubercles produced by *M. tuberculosis*.

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INFECTION WITH STREPTOBACILLUS MONILI-FORMIS. Occasional outbreaks.

Acute.

Chronic, with tendency to recovery.

Recovered animals have acquired immunity, but may be carriers and thus continue to spread infection.

SYMPTOMS.—

Acute Type.—

Mouse looks ill.

Coat dull.

Conjunctivitis: Seropurulent discharge. Eyelids sometimes glued together.

Death in a few days.

Chronic Type.—

Emaciation.

Conjunctivitis and keratitis.

Polyarthritis: Deformity and ankylosis of affected joints.

Œdema and cyanosis of extremities.

Paralysis of hind legs: Occasionally caused from involvement of vertebral column.

Ulceration of feet in a few cases, with crusting but rarely gangrene.

Œdema and cyanosis of tail: Bulbous swellings.

Post-mortem.—

Acute Type.—No characteristic changes.

Chronic Type.—

Joints: Disintegration; thick caseous exudate

in advanced cases.

Lymph glands: Regional and abdominal glands enlarged.

Spleen: Moderately enlarged; may contain a

small abscess.

Pericarditis: Frequent.

CAUSAL ORGANISMS.—

Streptobacillus moniliformis (Actinomyces muris) isolated from joint material, internal organs, and blood.

May be found in association with a *pleuropneumonia-like* organism, which by itself is relatively avirulent (Klieneberger).

Infection transmitted by direct contact and cohabitation and possibly by bites.

N.B.—Pleuropneumonia-like organisms are filterable, and are important as they may be discovered in laboratory mice and complicate research on filterable viruses. Infectivity appears to be low. Pneumonia can be produced, and also a variety of nervous signs and symptoms, choreiform in type.

"Rolling" phenomenon is observed in perhaps 10 per cent. of mice inoculated intracerebrally with other viruses. Death sometimes within twenty-four hours. The organisms appear

as granules, globules, and fine filaments.

INFECTIOUS CATARRH OF MICE or "MOUSE CATARRH." Sporadic and epizootic types. Infrequent. Tends to be chronic.

SYMPTOMS.—Variable. May include:—

Wheezing.

Intermittent "chattering."

Rhinitis, without visible nasal discharge.

Abnormal noises may be heard only when mouse apparently asleep; they stop abruptly when it is disturbed.

Ruffing and loss of hair.

Scabby skin.

Abrasions round the ear.

Respiratory distress.

Conjunctivitis: Infrequent.

MICE

POST-MORTEM.—

Rhinitis
Otitis media
Each in 30 to 50 per cent of cases.
Pneumonia.

Causal Organisms.—Small Gram-negative coccobacillary bodies (0.3 to 0.4 μ in diameter) regularly found in exudates. Possibly related to pleuropneumonia-like organisms referred to in previous section.

CONTROL.—

Mice showing symptoms killed.

Infection not necessarily serious; as a rule contacts need only be kept under observation.

PYOGENIC INFECTIONS. Pyæmic and suppurative lesions: Frequent.

Abscesses.—

Subcutaneous, e.g., about head and neck.

Heart.

Lungs.

CAUSAL ORGANISMS.—Wide variety.

Staphylococcus aureus: Commonest finding. Hæmolytic and non-hæmolytic streptococci.

Epizootic also described, due to hæmolytic streptococcus.

SYMPTOMS.—

Illness.

Eyelids adherent.

Hair roughened.

Breathing rapid.

Post-mortem.—Spleen: Enlarged, with pin-point abscesses throughout.

HEPATITIS DUE TO BACILLUS PILIFORMIS or TYZZER'S DISEASE. More common than is generally believed. First described in Japanese waltzing mice. May be serious, with diarrhœa and death. Under ordinary conditions the common laboratory mouse may be a carrier and not a case.

Injection with experimental material may light up infection.

Post-mortem.—Liver enlarged. Yellow necrotic lesions which may be confluent.

Causal Organism.—Bacillus piliformis, which is a long, filamentous, Gram-negative bacillus. Present in gut and lymphatic glands.

Control.—Must be energetic to prevent widespread epizootic. Slaughter or isolation of contacts. Sterilisation of cages, etc.

INFECTIOUS ECTROMELIA or "MOUSE-POX." A fairly common epizootic due to a filterable virus. Two types:—

Acute or abdominal.

Chronic or cutaneous, with recovery as a rule.

Recovered animals immune, but may be carriers and cause further outbreaks.

Wild mice may also be source of infection.

SYMPTOMS.—

Acute Type.—Recognition difficult.
Mouse looks ill.
Coat ruffled and dull.
No skin lesions.

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Death: Sometimes in four hours. (Mortality rate may be 80 to 90 per cent.)

Chronic Type.—

Skin lesions almost pathognomonic.

Enlargement of one foot—usually a hind foot—due to œdema. Followed by exudation of serous fluid and scab formation.

Gangrene of a toe or whole foot may follow.

Usually recovery unless spread to other sites, e.g.:

Other feet
Tail
Skin around mouth
Skin over body

If these involved, death results.

Differential Diagnosis.—From senile gangrene, which, as name implies, occurs in old mice; is not associated with œdema in early stages, and usually causes no general signs of illness.

Post-mortem.—

Acute Type.—

Generalised congestion.

Occasional small hæmorrhages.

Excess of peritoneal, pleural, and pericardial fluid.

Liver Spleen Diffuse necrosis.

Eosinophil inclusion bodies in cells of epidermis, intestine, and pancreas.

Chronic Type.—

Skin lesions, as described.

Internal organs: More extensive changes than in acute form.

Causal Organism.—Filterable virus isolated from internal organs, ædema fluids, and blood. (Ectromelia is closely related to vaccinia and the other pock diseases.)

VIRUS DISEASES

(Other than Infectious Ectromelia)

LYMPHOCYTIC CHORIOMENINGITIS. Often latent.

Symptoms and Signs.—Variable, but may include:—
Spastic convulsions of hind legs after mouse lifted by tail.
Animal may drag itself along table with its fore legs.
Conjunctivitis which may result in partially closed eyes.
Hunched back.
Loss of weight.

The virus is infective for man.

SPONTANEOUS ENCEPHALOMYELITIS. Virus often latent.

Disease recognised possibly by flaccid paralysis of hind legs without other apparent signs. Death as a rule.

VIRUS PNEUMONIA. Many types described.

A nuisance in studies of other virus infections. Latent infections may be activated when some other material is inoculated. (Investigations may also be complicated by infection with *Streptobacillus moniliformis*, which is discussed in a previous section.)

MISCELLANEOUS INFECTIONS, ETC.

ringworm and FAVUS. Not uncommon. Several varieties. (Sometimes these and other skin conditions associated with loss of hair are classified loosely as Mange.)

May spread as an epizootic.

Bald patches with inflammatory thickening and scaliness of the skin.

Fungi found inside and outside the hairs.

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CONTROL.—

Iodine in potassium iodide, Whitfield's ointment, etc.

In bad epizootic, which is exceptional, may have to slaughter animals.

Minor outbreaks, which are readily controlled but difficult to eradicate completely, are more common.

Care in handling, as man readily infected. Infections of nail-bed in man specially difficult to treat.

Washing hands in dettol reduces incidence of infection.

WORMS AND OTHER PARASITES.

Mice liable to infection: No symptoms as a rule.

If conditions not hygienic, ill-health or death.

Mostly nematodes and the cysticercus stage of cestodes.

Cysts of the cat tapeworm, Tænia crassicollis, are very common.

Cysts may be found in liver, usually singly but occasionally in pairs.

If worm dies, cyst may fibrose, become infected, and give rise to fatal septicæmia.

BARTONELLA, EPERYTHROZOON, GRAHAMELLA.

Three types of organisms which parasitise red blood cells. Although mentioned in literature, none produces clinical disease under natural conditions. Unimportant.

TUMOURS. Not uncommon in certain strains of mice. Particularly mammary carcinoma.

CONTROL OF INFECTIONS IN MICE

Intelligent, well-trained, and thoroughly reliable assistants are essential.

Unhealthy and *overcrowded* conditions avoided. Emphasis on prevention—not on treatment.

Remember that carriers, which are often mice left behind after an epizootic, may be responsible for spread.

(Latent infections in carriers are sometimes activated by injection of mucin or some other experimental procedure.)

Post-mortem examinations, with cultures, of all carcases the ideal procedure.

Cultures to be made shortly after death. Otherwise the causal bacteria may be outgrown by secondary invaders from gut.

Cages or Boxes.—Kept in rodent-proof, light, well-ventilated room.

Frequent disinfection of cages and hosing down of racks and floors. Lysol or cresols harmful.

Racks spacious—no contact of individual cages. Metal racks and boxes preferable to wooden ones; some workers prefer glass jars to boxes.

Temperature and Humidity.—Regulated. Sudden variations harmful. Thermostatic control desirable.

Bedding.—Best stored in bins or metal containers in special rooms protected from vermin and stray rodents.

Must be dry, and is put only into cages which have been dried.

Sterilised soft wood shavings most satisfactory.

(Teak, mahogany, and other hard woods harmful due to phenolic substances in resinous woods.)

Soiled bedding removed twice weekly and burned.

Food and Water.—Storage food-bins or metal containers should be kept in special rooms protected from vermin.

Nutritionally complete diet.

Regular feeding.

Water should always be provided and must be clean and fresh.

Water bottles, like other utensils, sterilised, preferably by steam.

Isolation of New Stock.—Not always feasible, but materially reduces risk of introducing infection into mouse rooms.

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Isolation for three weeks; stock distributed in cages containing not more than four to six mice each.

Not a good practice to mix mice from different colonies.

Newcomers to a given population may acquire infection from, or give it to, the original stock, and light up an epizootic which may finally involve both groups.

Immediate Destruction of Ailing Animals.—Always to be considered.

Burn carcases after autopsy.

Destruction of all Animals in Batch.—May be necessary if epizootic extensive.

Chemotherapy.—Limited usefulness in controlling certain infections in valuable stocks.

RATS

Domesticated varieties of the Norway or brown rat (Rattus norvegicus) are healthy animals if well fed and housed.

Intestinal and respiratory infections occur, however, especially in albino stock.

INTESTINAL INFECTIONS. Not uncommon. Often called Paratyphoid Disease.

Salmonella group of organisms, e.g., S. enteritidis, may cause illness as in other laboratory animals.

Acute Type.—

Loss of weight.
Scabs about nose.
Pallor of ears.
Possibly diarrhæa.
Usually severe anæmia.
High mortality.

Chronic Type.—

More common.

Altogether milder.

Infected animals may look and appear well for long periods, suggesting a carrier condition. Carriers pass on the disease to other animals.

Post-mortem.—

Appearances in *acute* type resemble those described for mice and other animals. Changes may include the following:—

Spleen: Enlarged and congested. Multiple small yellow-grey necrotic foci and hæmorrhages.

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Liver swollen, pale, and friable, and may also show areas of necrosis.

Intestines inflamed and contents blood-stained.

Lymphatic tissue in gut inflamed and may show some ulceration.

Lungs pneumonic, with some hæmorrhages.

Certain lymph glands enlarged, especially thoracic group.

In *chronic* type, lesions mostly restricted to intestines, which may be thickened and ulcerated in region of cæcum. Lymph tissue in ileum may show swelling, usually without ulceration.

Local mesenteric glands often enlarged and cystic.

Spleen swollen and congested.

Liver Lungs Usually normal.

Causal Organisms.—Salmonella group, e.g., S. enteritidis (B. gaertner) and S. typhimurium (B. ærtrycke).

CONTROL.

Infected wild rats and mice may convey infection to food-stuffs and should be excluded.

As mentioned above, laboratory and other rats may show no obvious signs of infection, but serve as *carriers*. It should be remembered also that carrier condition may be set up by "virus" poisons, which consist of living cultures of Salmonella, mixed with a palatable preparation.

Vaccines of Salmonella strains suggested. Little value and do not justify expense and trouble. Search for carriers with view to exclusion has been tried, but is not recommended.

As with other laboratory animals, careful attention to cleanliness and other hygienic measures.

Proper feeding also important.

RESPIRATORY INFECTIONS. Include Pneumonia, Broncho-pneumonia, and Middle Ear Disease and Labyrinthitis.

Pneumonia.—Common morbid condition of adult rats.

May be induced by sudden variations in temperature.

Is acute and rapidly fatal.

Associated with grey areas of lung, possibly with some hæmorrhages.

Suppurative otitis may be present as complication.

CAUSAL ORGANISMS.—

Hæmolytic streptococci readily isolated from lungs.

Hæmophilus bronchisepticus

Diphtheroids Sometimes isolated also.

Streptobacillus moniliformis

Broncho-pneumonia.—Described by Klieneberger and Steabben.

Chronic infection.

May cause little disturbance of structure or function.

Appetite and movement may be unimpaired.

POST-MORTEM.—

Lungs: Possibly grey translucent nodules and hæmorrhages.

Nothing abnormal may be detected.

CAUSAL ORGANISMS.—

Believed to be S. moniliformis associated with pleuropneumonia-like organisms.

Special cultural methods necessary for isolation; ordinary cultures from lungs are typically sterile.

N.B.—Fifty per cent of wild and domesticated rats said to harbour the Streptobacillus in nasopharynx, where it produces no symptoms.

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Middle Ear Disease and Labyrinthitis.—Considered together for convenience.

Suppurative otitis in which inflammation has extended from middle ear to labyrinth and thus caused symptoms.

In very many symptomless cases, middle ear alone infected. This is itself a sequela and extension of inflammation of upper respiratory tract—essentially similar to infections commonly observed in rabbits.

Rats under three months apparently immune.

Older rats: Variable incidence in different colonies (up to 50 per cent at post-mortem).

Infection may be bilateral, the most obvious sign being greyish-green pus of varying consistency.

Approximately 4 per cent of rats showing infection in tympanic cavity have associated labyrinthitis.

May be further extension to petrous bone and auditory nerve.

SYMPTOMS.—

Tilting of head.

Animal runs in a curve.

Sometimes staggering.

Falling and difficulty in getting up.

When suspended by tail, rat rotates rapidly. On account of this phenomenon, rats with labyrinthitis sometimes referred to as "twisters."

CAUSAL ORGANISMS.—

Probably ordinary pathogens.

Possibly S. moniliformis and pleuropneumonia-like organisms, the exact rôle of which has not yet been determined.

CONTROL.—

Selective breeding of resistant strains of rats suggested. Chemotherapy might be worth while when more known about bacteriology of condition locally.

PURULENT LYMPHADENITIS and POLYARTHRITIS.

-Uncommon. Described mainly by British workers.

CAUSAL ORGANISM.—A pleuropneumonia-like organism, and not a filterable virus, as was first thought.

MANGE, "RAT SCAB," or "RAT SCABIES." Due to a species of mite (Notoëdres).

Grey warty lesions:-

Root of tail.

Ears.

Nose.

This condition is transmitted to man.

CONTROL.—

Animals examined twice weekly for fresh cases.

Local treatment (benzyl benzoate, dimethyl-thianthrene, gammexane preparations such as Lorexane, Tetmosol, etc.). *Cf.* mange in rabbits.

Easily cured.

WORMS.

Cysts of the Cat Tapeworm (Tænia crassicollis).—

Widely distributed.

Ivory-white cysts: Liver. Mesentery.

Other Parasitic Worms.—

Many species.

Less common amongst laboratory rats than wild rats.

PRINCIPLES OF CONTROL OF INFECTIONS

As for other laboratory animals (see previous sections).

HAMSTERS

Hardy. Usual laboratory infections relatively rare.

SALMONELLA INFECTIONS. Outbreaks occur sometimes.

SYMPTOMS.—

Diarrhœa.

Lack of appetite.

Ruffed hair.

PNEUMONIA. Infectious type has been reported.

SYMPTOMS.—

Catarrh.

Emaciation.

Loss of appetite.

CONTROL.—Destruction of affected and contact animals (see sections on other animals).

LEISHMANIA INFECTION. Common.

MANGE. Common. Due to burrowing mite, Notoëdres notoëdres.

SYMPTOMS.—

Ears and other parts of body affected.

May lead to much scabbing.

CONTROL.—Usual anti-mange remedies.

VIRUS DISEASES. A latent virus pneumonia recorded. Incidence unknown.

FERRETS

COCCIDIOSIS. Common. Chiefly young ferrets under four to five months.

SYMPTOMS.—

Diarrhœa, with mucous or tarry stools (melæna). High mortality.

Post-mortem.—

Intestines inflamed—often blue-green.

Demonstration of oöcysts in smears of intestinal contents.

CONTROL.—

Bitch ferrets examined for carrier condition before breeding.

Ferrets, and especially weaned stock, kept in cages with wire floors and shelf for tray underneath.

As soon as young ferrets weaned, litters should be segregated from the bitches.

Chemotherapy with sulpha drugs might be useful and is worthy of trial.

DISTEMPER. Due to virus of canine distemper. This is the most serious disease of the ferret.

Highly infectious: May be 100 per cent case incidence, with 90 to 95 per cent mortality.

Incubation period: Eight to twelve days.

Symptoms.—

Ferret sleepy and curls up; lack of interest in surroundings.

Eyes and nose: Watery discharge, becoming purulent.

Eyelids: Stuck together.

Mouth: Vesicles develop about eighth day.
Become pustular.

Skin of abdomen and feet congested.

Loss of appetite.

Loss of weight.

Weakness progressive.

Epileptiform convulsions in a few cases. These begin as muscular twitchings and tremor, which may develop into violent and generalised convulsive movements associated with screaming. (Convulsions are not always due to distemper virus.)

Death from pneumonia (in many cases probably virus pneumonia).

CONTROL.—By active immunisation.

Formolised virus vaccine: Preferably two doses spaced by ten days.

Living virus vaccine: Ten days later.

Every precaution taken to prevent introduction of virus into ferrets' quarters.

Unauthorised visitors excluded.

Attendants and authorised visitors should wear waterproof coats and boots (and, where ferrets are to be handled, gloves) which are washed down with disinfectant solution before entry.

Trays or troughs of disinfectant placed at doors. (Entrants step on trays.)

As further safeguard these doors may be kept locked.

When distemper develops, treatment unsatisfactory.

Affected ferrets should be killed as soon as symptoms appear.

Strict isolation of remainder of stock necessary.

Disinfection of cages, utensils, etc., must be thorough.

HARD PAD DISEASE. Due to a virus which appears to be a variant of the canine distemper virus; the two viruses are related antigenically.

Incubation period: Up to thirty-five days, and usually seventeen to twenty-three days.

SYMPTOMS.—

Very similar to distemper.

Pads of feet become enlarged and hard due to hyperkeratosis.

Discharges less purulent than in distemper.

CONTROL.—As for distemper. Research still in progress.

INFLUENZA. Due to influenza viruses. Resembles mild distemper, but no vesicles.

SYMPTOMS.—

Sneezing.

Nose Thin watery discharge. Dries. Scabs form around nostrils. Nose may be blocked.

Mortality usually low, but acute epizootic may appear, with high mortality.

Blood-stained nasal discharge and black crusts. Hæmolytic streptococci isolated readily from most cases.

CONTROL.—Measures similar to those described in previous section on Distemper.

OTHER VIRUS DISEASES.

A number have been described, epizootic in type and associated with high fatality rate.

Secondary invasion may occur with other bacteria, notably hæmolytic streptococci.

streptococcal infections. May complicate influenza and other diseases (see above). May also occur independently as sporadic cases or as epizootic, difficult to diagnose.

SYMPTOMS AND SIGNS.—

Pneumonia, septicæmia, widespread hæmorrhages, large spleen.

Lesions around mouth and nose, simulating distemper. Group C streptococci are most commonly implicated.

ABSCESSES. Sometimes develop in neck. May involve salivary glands and deeper tissues, including bone.

CAUSAL ORGANISM.—Staphylococcus aureus. Site of entry probably some breach of skin or mucous membrane.

TUBERCULOSIS. Observed in ferrets fed on unheated milk of tuberculous cows; it is then of bovine type.

Avian infection also occurs and is likewise of alimentary origin.

Tuberculosis is a nuisance to those responsible for production of distemper virus vaccines or engaged in study of other infections.

SYMPTOMS AND POST-MORTEM.—

The *bovine* type becomes generalised, with the macroscopic signs, symptoms and post-mortem appearances described for guinea-pigs.

Minor degrees of infection give rise to little or no abnormality, and diagnosis may be difficult.

Avian infection is associated with minimal naked-eye lesions, possibly only an enlarged spleen, with no tubercles.

CONTROL.—

Boiling or pasteurisation of milk.

Supervision (and, where necessary, heat treatment) of other food-stuffs which might contain tubercle bacilli.

FOOT-ROT. Common. Caused by a mite.

SYMPTOMS AND SIGNS.—

Swelling and scab formation of feet, mainly between toes. Claws may slough off if disease untreated.

CONTROL.—

Attention to cleanliness and other hygienic measures.

Treatment not difficult in early stages.

Scabs may be softened by immersion of lesions in warm water. Cleaning up of affected parts thus facilitated.

Iodine, kerosene, or xylol then applied.

Benzyl benzoate or dimethyl-thianthrene may be tried.

SCABIES. Caused by a mite. Back and tail affected.

CONTROL.

Successful.

After removal of crusts, iodine, kerosene, xylol, benzyl benzoate, and sulphur ointment have all proved useful.

TOXOPLASMOSIS. Infection generalised.

CAUSAL ORGANISM.—T. laidlawi.

CONTROL OF INFECTIONS IN FERRETS

General principles as for animals previously considered, viz.:—

Cleanliness.

Disinfection of cages.

Nutritious diet free from contamination with M. tuberculosis. Also, ferrets acquired from dealers or some other laboratory

should be isolated for at least thirty days before use.

MONKEYS

Usually healthy in captivity, after acclimatisation. Good feeding and housing essential.

PNEUMONIA. Sporadic cases and epizootics, especially during winter months.

TUBERCULOSIS. Commonest infection of monkeys in capivity. Human and bovine types.

Frequently gastro-intestinal form.

PREVENTION.—

Give only boiled milk.

Improve hygiene and housing.

Eliminate all reactors to tuberculin test. Retest at six-month intervals for two years, thereafter at yearly intervals.

PSEUDO-TUBERCULOSIS. Confused with tuberculosis. Infection probably acquired from rodents.

RICKETS. Young monkeys very liable to suffer. Attention to diet.

VIRUS DISEASES. Lymphocytic choriomeningitis and "B" virus reported. Incidence unknown. Unimportant until stimulation by other infections.

CATS

Worms and also various diseases of unknown ætiology omitted.

CAT DISTEMPER (Nasal Catarrh, Influenza).

Epizootic, chiefly respiratory.

Mainly upper air passages.

Appears to be caused by virus entirely different from that infecting dogs.

Acute.

Chronic: Convalescence may take one to two months.

SYMPTOMS.—

Sneezing.

Slight fever.

Nasal and eye discharge: Becomes purulent.

Loss of appetite.

Loss of weight and condition.

Staring coat.

Exaggerated "cat smell."

Constipation: Usual.

POST-MORTEM.—

Often pneumonia, with bilateral purulent pleurisy. Various organisms isolated: Secondary invaders (commonly Staphylococcus aureus).

CONTROL.—

Isolation.

Good nursing.

CATS

INFECTIOUS ENTERITIS (Feline Enteritis). Highly infectious disease of young cats. Due to virus.

Total cat population of district may be wiped out. All feline species affected, including leopards and cheetahs. Incubation period: One to five days.

Symptoms.—

Sudden onset and rapid course.

Almost always fatal.

High temperature (106° F.).

Loss of appetite.

Vomiting and pain in abdomen.

Great depression and no interest in surroundings.

Cat lies on abdomen with hind legs extended back, fore legs drawn in, and head bent down.

Eyes sunken.

Death possibly before symptoms noticed.

POST-MORTEM.—

Thorax usually normal.

Jejunum inflamed, faint pink to rich purple.

Mesenteric glands: Enlarged and hæmorrhagic.

Peritonitis occurs in some outbreaks. When purulent, probably due to hæmolytic streptococcus.

DIAGNOSIS.—Blood smears: Decrease of leucocytes (to nil in some cases).

CONTROL.—

Can use formolised spleen vaccine (from spleens of fatalities).

Eliminate fleas: D.D.T., etc.

Isolation also helps.

ULCERATIVE STOMATITIS. Ulceration of gums, with much erosion. Due to virus.

STREPTOCOCCAL TONSILLITIS. Fairly common.

SALMONELLA INFECTIONS. Fatal infections and also carrier condition reported.

Cats constantly eat rats and mice, and yet infection is apparently less common than in dogs.

TUBERCULOSIS. Cats more susceptible to bovine bacillus than to human.

Sometimes generalised infection (Siamese said to be the most susceptible breed).

Source likely to be milk.

Organism often demonstrated in nasal discharge of cases of "catarrh."

FUNGOUS INFECTIONS. Parasitic skin diseases more common in cats than in dogs.

Various species of Microsporum recognised.

DOGS

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DOGS

SALMONELLA INFECTIONS. Acute intestinal disease sometimes reported.

SPIROCHÆTAL INFECTIONS. Two varieties:—

1. Due to Leptospira icterohæmorrhagiæ.

Disease known as "Yellows," pathologically similar to Weil's disease in man.

Dog usually infected by rat-contaminated food.

Many dogs are healthy urinary carriers for a brief period after clinical infection, with risk to other dogs and to man.

SYMPTOMS.—

High fever.

Vomiting.

Hæmorrhages: Nose, gum, rectal (possibly from intussusception).

After five to six days there is—

Jaundice: A prominent symptom (wherefore "Yellows").

Urine: Bright yellow.

Coma.

Toxic convulsions.

Mortality rate is high.

POST-MORTEM.—

Icterus and petechial hæmorrhages: Most organs. Lungs: "Butterfly" appearance (due to hæmorrhages). Small intestines: Intussusception.

DIAGNOSIS.—

Scarification of depilated guinea-pig skin, followed by "rubbing in" a few drops of liver emulsion from dog; characteristic infection usually produced, and diagnosis confirmed.

Control.—Vaccination with phenolised culture: Two doses at interval of four weeks.

For treatment:

Hyperimmune serum.

Penicillin not very effective. Streptomycin and aureomycin more hopeful.

High liquid intake.

Remember that work with leptospirosis is a hazard to laboratory workers. The use of gloves and also of goggles recommended.

2. Due to L. canicola, which causes nephritis, not jaundice. Infection also called "Dog Typhus" or "Stuttgart Disease."

SYMPTOMS AND SIGNS.—

Often no fever at onset.

Vomiting.

Intense thirst.

Anorexia.

Emaciation and dehydration.

Conjunctivæ: Dirty red.

Stomatitis, with ulcers on tongue and lips.

Uræmic smell from breath.

Abdomen tender.

Urine: Pale.

Recovery after two weeks, or death, usually after one week. Case mortality rate is high in clinically recognisable cases.

If recovery, many dogs are left with a chronic nephritis.

Healthy carriers not uncommon. (In surveys there is a high positive serological rate.)

Post-mortem.—

Gastro-enteritis.

Kidneys: Congestion, etc.

CONTROL.

Vaccine recently introduced for prevention.

· DOGS

Penicillin and fluids for treatment.

After first week of infection, streptomycin of value, but not penicillin.

BARTONELLOSIS. Organisms of the genus *Bartonella* occur widely. They parasitise blood-cells.

Transmitted by blood-sucking arthropods, usually lice.

Unimportant unless disturbance of health, when severe and sometimes fatal anæmia may be caused.

FUNGOUS INFECTIONS. Acquired possibly from street animals.

May spread easily in kennels.

Usually limited to skin.

Small, circular, thickened, scaly, bare patches.

Causal Organisms.—Belong to genera Microsporum and Trichophyton.

Control.—Iodide or 5 per cent alcoholic solution of salicylic acid.

DOG DISTEMPER. Acute generalised catarrhal disease with a diphasic fever.

Highly contagious and often fatal.

Incubation period: Four to seven days.

SYMPTOMS.—

Watery nasal discharge becoming purulent and bloodstained.

Ocular discharge.

Diarrhœa may predominate.

Interstitial pneumonia (due to virus) slight, but often followed by secondary broncho-pneumonia (B. coli, Salmonella, H. bronchisepticus, etc.).

Nervous complications: Usually terminal or after apparent recovery. Consist of chorea and/or epileptiform convulsions.

CAUSAL ORGANISM.—Virus.

CONTROL.—

Active immunisation with canine distemper prophylactics comprising:—

Vaccine: Inactivated virus preparation.

Virus: Dried preparation of living virus, reconstituted immediately before use with special

diluting fluid.

Serum: Obtained from dogs hyperimmunised against

distemper virus.

Two methods of immunisation, viz.:

Method 1, or "vaccine-virus."

Vaccine followed by virus ten to twenty-one days later.

Method 2, or "virus-serum."

Virus, followed by serum one to twenty-four hours later.

Method I is preferable and should be used whenever practicable.

Very young puppies can be protected over a short period of risk by injecting 10 c.c. of serum.

TREATMENT.—

Twenty to 50 c.c. of hyperimmune serum.

B. coli anti-serum
Anti-bronchisepticus serum
Sulphanilamide
Penicillin
To control secondary
infection.

Also symptomatic treatment and good nursing.

DOGS 53

HARD PAD DISEASE. Due to a virus related to distemper virus.

SYMPTOMS.—

Catarrh from eyes and nose: Less than in distemper.

Is rarely purulent, and is often absent.

Fever: Monophasic. Often absent in early stages.

Diarrhœa: Usually present for one week before any other symptoms. May be severe and persistent.

Photophobia.

Conjunctivæ: Congested.

Pads of feet show hyperkeratosis just before or at onset of nervous manifestations, e.g., chattering of teeth (spasm of masseters), convulsions, screaming, walking in circles.

Dyspnœa: Often severe, due to pulmonary œdema (possibly neurogenic origin).

Dehydration: Often marked.

TREATMENT.—

Hyperimmune serum: 1 c.c. per lb. body-weight, repeated after four days.

Protein hydrolysate where anorexia is complete.

Aneurine hydrochloride: 25 mgm. intramuscularly for spasm of masseters.

Glucose saline, etc., for dehydration.

Aspirin and small doses of barbiturates.

No sulpha drugs.

FOX ENCEPHALITIS or CONTAGIOUS HEPATITIS.

A virus disease of dogs and foxes.

Affects dogs of all ages, but highest death-rate in very young puppies.

Incubation period: Variable, but usually short.

SYMPTOMS.—

Fever, apathy, coma, and death (often within twenty-four hours).

If the dog lives two or more days:—

Anorexia.

Thirst.

Vomiting.

Hæmorrhagic diarrhæa.

Fever up to 106°.

Violent convulsions.

Coma and death.

Post-mortem.—

Liver: Swollen and hæmorrhagic, with fibrin shreds between lobes.

Gall-bladder: Walls thickened.

Peritoneal and pleural effusion.

Hæmorrhages in most organs, including brain.

A liver section will show numerous nuclear inclusions.

ORAL PAPILLOMATOSIS. Highly contagious. Spread by contact. Causes serious inconvenience to the animal.

MISCELLANEOUS INFECTIONS

Coccidiosis, due to one or more species of coccidia.

Other **Protozoan Infections**, including those due to *Toxoplasma gondii* and *Babesia canis*. The latter organism, which is a parasite of the erythrocyte in warm countries, is transmitted by several species of tick.

It may cause an infection with high fever, anæmia, jaundice, and hæmoglobinuria, thus resembling leptospirosis.

Treated with "Piropary."

Infections with various Parasitic Worms (cestodes and nematodes).

PIGEONS

SALMONELLA PARATYPHOID. INFECTION or

Common.

SYMPTOMS.—

Acute (young birds as a rule).

Death within forty-eight hours.

Chronic (disease more prolonged and varied in older birds than in the young).

Shaking of head.

Twisting of neck.

Discoloration of eyes.

Laboured respiration Frequent.

Diarrhœa

Joints of legs and wings: Residual arthritis sometimes, if bird recovers.

Carrier condition may become established.

Post-mortem.—

Intestines?

- Congested. Kidneys

Congested, possibly bile-stained with small Liver:

areas of necrosis.

Sometimes enlarged.

Lungs: Often pneumonia.

Organism.—A variant of S. typhimurium (B. ærtrycke), designated S. typhimurium var. Storrs.

CONTROL.—

Attention to hygiene.

Sterilisation of cages, etc.

Repeated agglutination tests for detection and elimination of carriers.

SWINE ERYSIPELAS. Pigeons very susceptible.

SYMPTOMS.—

Loss of appetite.

Ruffled feathers.

Head sinks into neck.

Death: In twenty-four to forty-eight hours, or earlier.

Smears and cultures from bone marrow will confirm infection.

CAUSAL ORGANISM.—Erysipelothrix rhusiopathiæ.

CONTROL.—Cages sterilised before other pigeons introduced.

PSITTACOSIS (Ornithosis). A virus infection. Widely distributed in different parts of world.

SYMPTOMS.—

Indefinite. Not characteristic.

Death after first indication of infection.

Mortality high in young birds, possibly 50 per cent in some flocks.

Post-mortem (all dead pigeons should be examined).—

Emaciation.

Main appearance is inflammation of one or more serous membranes, especially fibrinous pericarditis and peritonitis.

Liver: Large and pale, with infarcts and necrotic areas.

Spleen: Enlarged.

As humans are susceptible, establish diagnosis:—

Smears of liver and exudates examined for virus.

Inoculate mice intraperitoneally with suitable pathological material.

CONTROL.

Isolation, etc.

Remember that apparently healthy pigeons may be carriers.

Complement-fixation tests useful in establishing diagnosis in infected birds.

In working with psittacosis virus, special care as considerable risk of human infection.

PIGEON-POX. Due to virus, related to fowl-pox.

Infection transmissible to pigeons only.

Common in certain lofts, and mainly affects birds at age of six to eight months.

SYMPTOMS AND SIGNS.—

Small nodules: Wattles, conjunctivæ, commissures of

beak, etc.

Crusts: After one week. Later, fall off.

Corneal infection: May accompany conjunctivitis.

Pharynx Yellowish-grey patches, which may be

Sides of tongue \(\) cheesy.

Loss of appetite. Loss of weight.

Lesions may extend to trachea and cause suffocation.

DIAGNOSIS.—

Made from appearance of lesions, and possibly as result of cutaneous inoculation of ground-up crusts into other pigeons.

Characteristic local lesion should develop.

Pigeon cannot close beak: Useful diagnostic sign.

Recovered pigeons are immune.

CONTROL.—

Active immunisation, which confers satisfactory protection. Vaccine painted, with a brush, over small area of defeathered breast at age of six months.

AVITAMINOSIS (A, B, D, and E). Pigeons specially liable to suffer. Influence on severity and spread of infections.

CANARIES

(After "U.F.A.W. Handbook," 1947, by kind permission of Dr. Jean Vintner.)

Infections introduced from stores, bird-rooms, or shows.

Survivors of infections are often carriers.

Carriers may remain well till subjected to special strains, e.g., mating, overcrowding, or moulting.

Isolate new birds in clean, disinfected cages for one month

or longer.

INFECTIOUS NECROSIS OF LIVER AND SPLEEN (Bird Fever, Paracholera, Pseudo-tuberculosis).

Highly infectious.

Incubation period: Three to five days.

Signs indefinite.

Death possibly within one week.

POST-MORTEM.—

Characteristic nodules in spleen and liver.

Diagnosis confirmed by bacteriological examination.

Causal Organism.—Pasteurella pseudo-tuberculosis rodentium. Is pathogenic to many animals, especially rodents.

PARATYPHOID (Typhoid, Contagious Enteritis, Septic Fever).

SYMPTOMS.—

Affected birds dull.

Ruffled feathers.

Diarrhœa.

Death may occur suddenly after convulsions.

POST-MORTEM.—

Liver Spleen Enlarged.

Intestines: Inflamed.

CAUSAL ORGANISMS.—

Common causes are Salmonella pullorum, which causes Bacillary White Diarrhœa of fowls.

S. gallinarum, which is associated with fowl typhoid.

Source of infection may be eggs from infected hens and food-stuffs made from diseased carcases.

S. typhimurium (B. ærtrycke) also found in paratyphoid.

All these organisms may be secondary invaders, complicating psittacosis.

If parrots, budgerigars, etc., in vicinity, psittacosis should be excluded.

STREPTOCOCCUS INFECTION.

SYMPTOMS.—

Birds dull and sleepy.

Diarrhœa.

Difficulty in breathing: Wheezing and squeaking.

POST-MORTEM.—

Liver

Lungs Spleen

Foci of inflammation and necrosis.

Other organs

Pleurisy Peritonitis

Possibly serofibrinous exudates.

CAUSAL ORGANISM.—Streptococcus, probably transmitted by red mite.

ASPERGILLOSIS. Common.

Acute.—Possibly death from infectious pneumonia in eight to twelve hours.

More Chronic.—

Difficulty in breathing, with wheezing, gurgling, and rattling noises.

Somnolence.

Thirst.

Pyrexia.

May become dull, weak, and emaciated.

POST-MORTEM.—

Whitish-yellow crusty coatings on tongue, palate, trachea, etc.

Lungs Air-sacs Caseous foci.

CAUSAL ORGANISM.—The fungus Aspergillus fumigatus.

CONTROL.—

Avoid mouldy food or litter.

Attention to hygiene.

Clean cages.

Exposure to direct sunlight and fresh air.

TUBERCULOSIS. Rare.

AVIAN POX. Not uncommon. Usually rapidly fatal. Three forms:—

- I. Wart-like lesions on comb, wattle, and face.
- 2. Diphtheritic lesions on membrane of mouth.
- 3. Localisation in nasal chambers (roup).

Diagnosis established by appearance, demonstration of inclusion bodies, and transmission experiments on suitable birds.

CAUSAL ORGANISM.—Virus of canary pox, pathogenic as a rule only for canaries and passerine birds.

CONTROL.—

Affected birds destroyed at once.

Isolation from possible carriers (newly purchased and wild birds).

Possibly active immunisation with live vaccine made from infecting strain.

PSITTACOSIS (Ornithosis). Source of infection usually infected parrots or parakeets.

Incubation period: Fifteen to sixty days.

SYMPTOMS.—

Listless.

Ruffled plumage.

Death possibly suddenly.

Duration of illness three to twenty days.

POST-MORTEM. As described for pigeons. CONTROL.

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